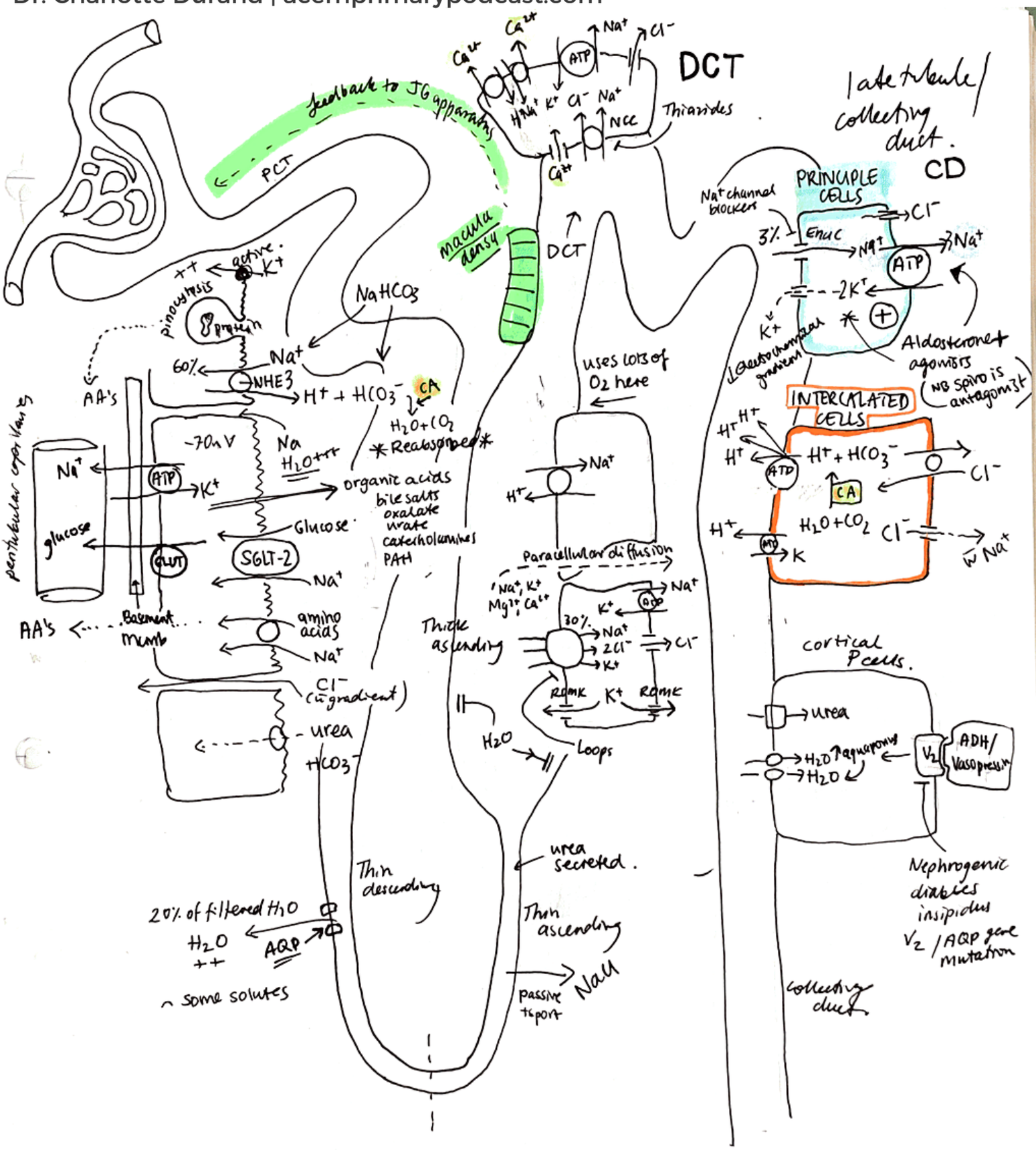


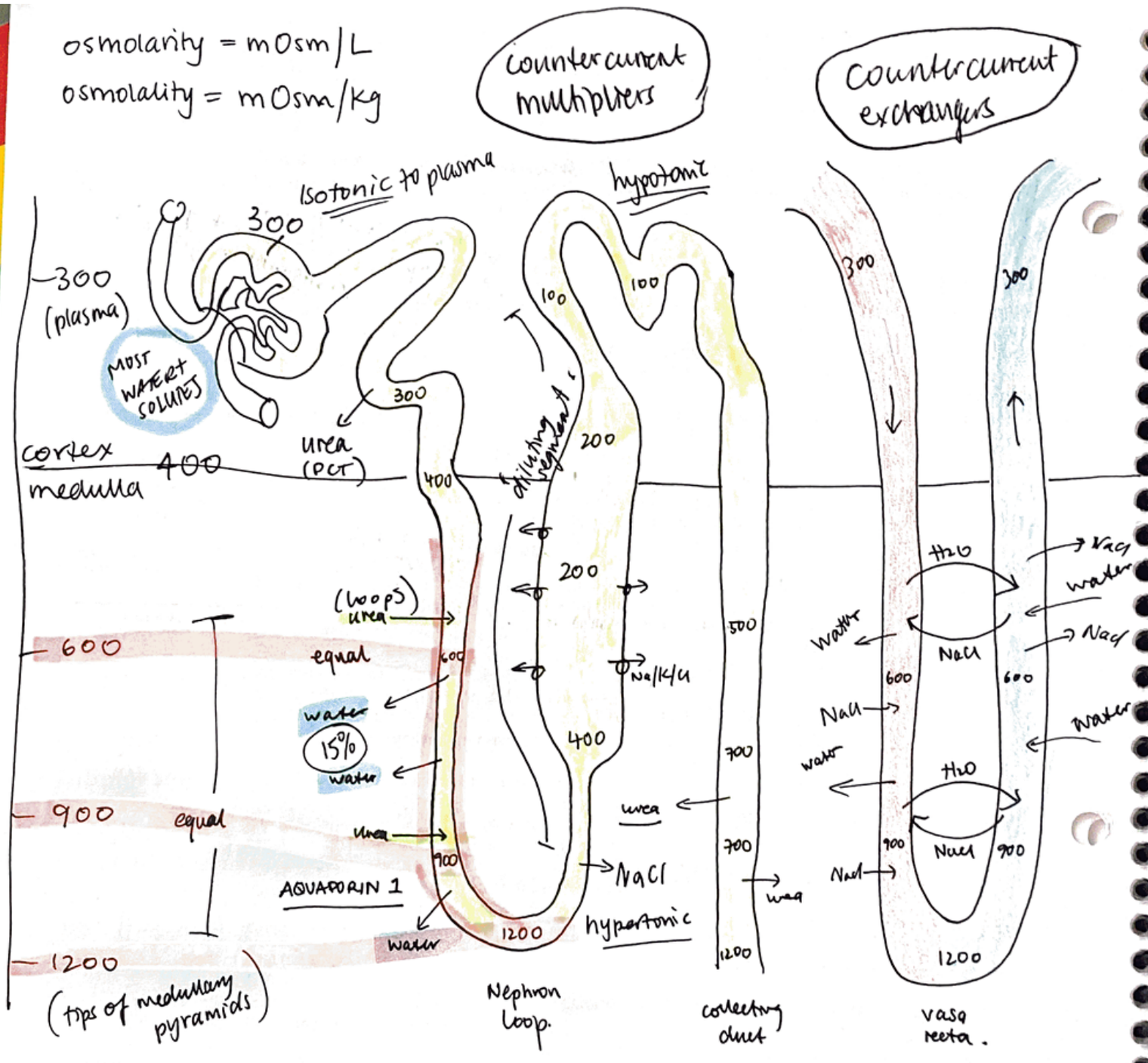
NB: Aldosterone

1. upregulates + activates Na/K/ATPase
2. ↑ ENaCs
3. ↑ Cl⁻ reabsorption via pull of Na⁺
4. Secretion of K⁺ into lumen
5. Effects on saliva, sweat, gut.
6. Stimulates Na^+/H^+ exchange in I cells

URINE

acidic pH
SG often 1.002 – 1.030
no protein
Urea 900 mg/dL & > creat.
Na < Serum Na
Cr > Serum
glucose 0.

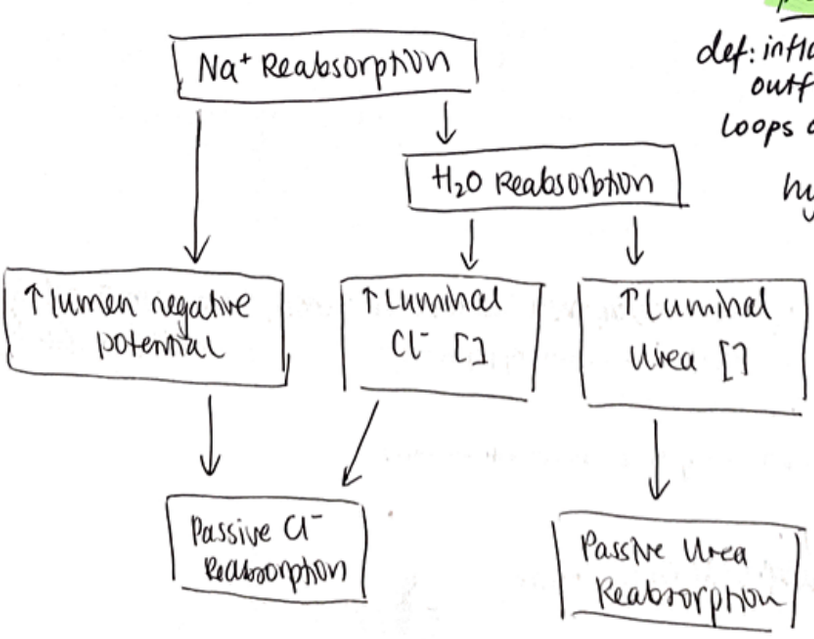
osmolarity = mOsm/L
osmolality = mOsm/kg



The Countercurrent Mechanism.

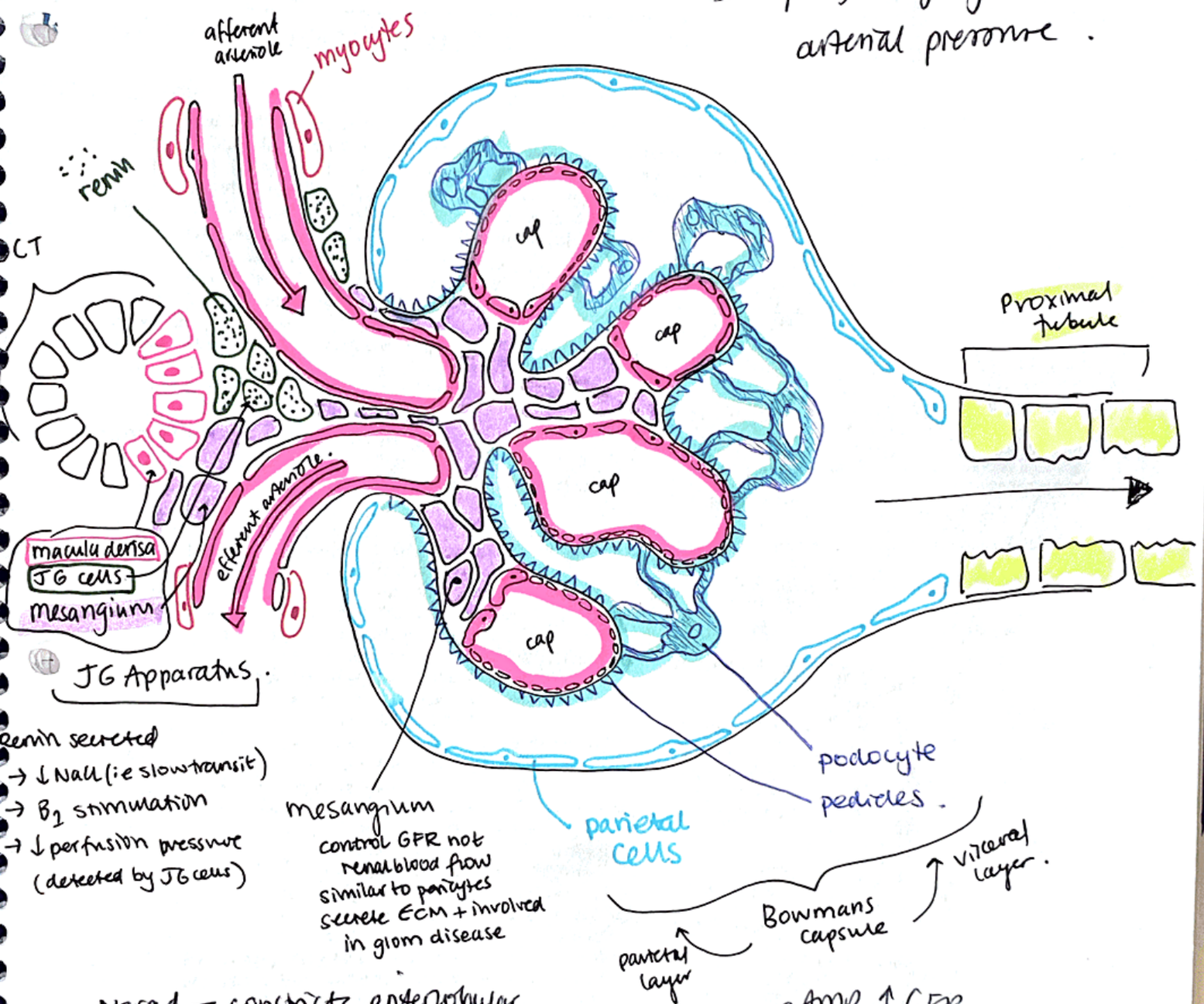
def: inflow runs parallel to, counter to & close to outflow for some distance.
Loops of Henle + vasa recta.

hypotonic fluid enters the medulla in the lumen of tubules & vasa recta.
water drawn out.
becomes hypertonic to equilibrate w interstitium.
when flowing back up it takes in water b/c interstitium is hyper.
pumps in thick ascending limb drive this by pumping out solutes but not water.



The Glomerulus

glomerular capillary pressure = 40% of systemic arterial pressure.



Renin secreted
→ ↓ NaCl (i.e slow transit)
→ B_1 stimulation
→ ↓ perfusion pressure (detected by JG cells)

Norad — constricts enterolobular & afferent vessels ↓GFR

cAMP ↑GFR

Dopamine causes renal vasodilation + natriuresis → ↑GFR.

ATII constricts afferent + efferent ⇒ ↓GFR.

PG's ↑ cortical flow, ↓ medullary flow

ANP ↑GFR via mesangial cells.

Ach = vasodilation.

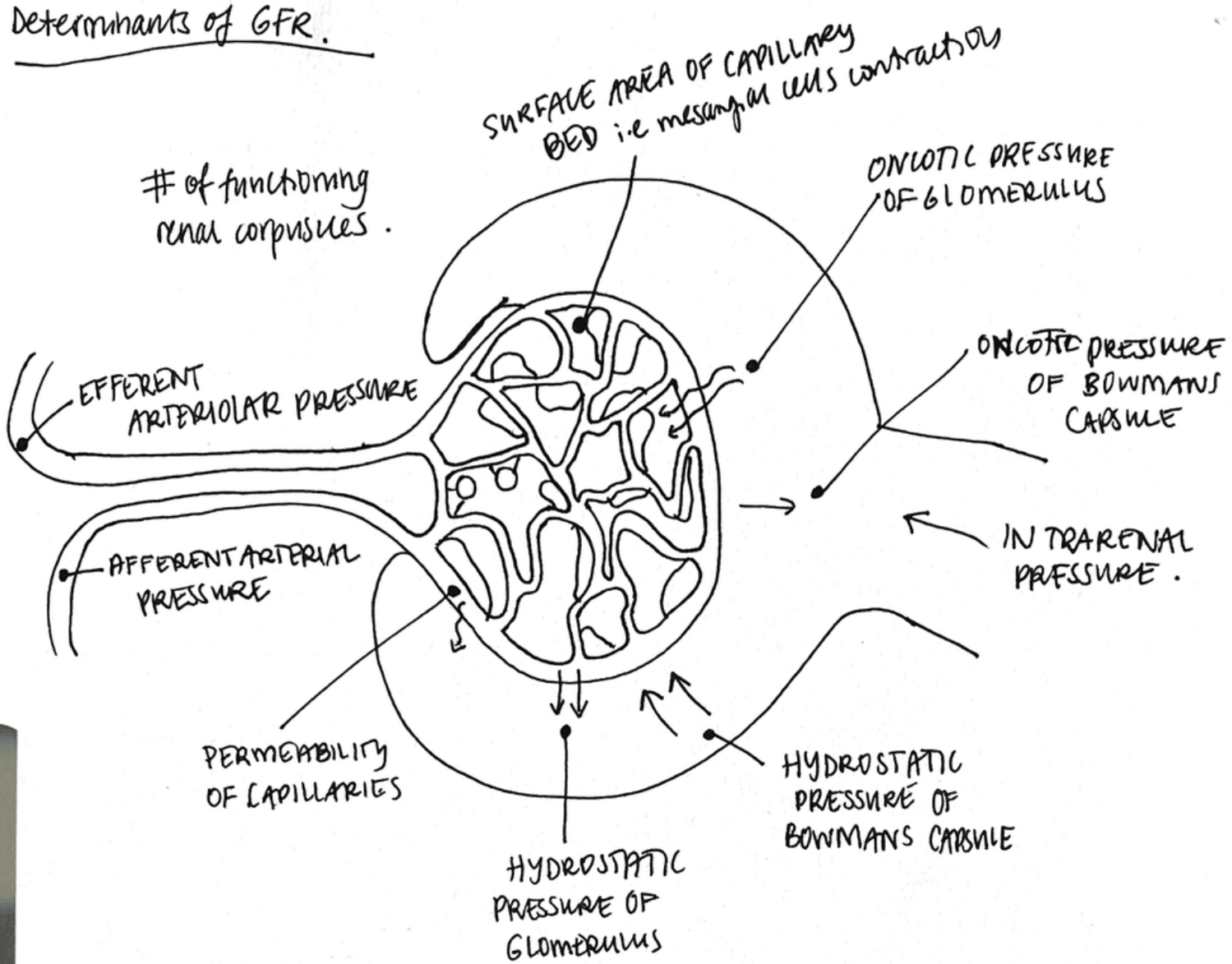
Histamine ↓GFR.

Kidneys get 23% ~~that~~ CO.

High protein diet → ↑ glomerular capillary pressure & ↑ renal blood flow

Exercise ↓ RPF — can fall to 25% of normal

Determinants of GFR.



PAH vs inulin

PAH used to measure renal blood flow
- ↳ filtered } 90% cleared.
- ↳ secreted. }
- ↳ [PAH] in renal vein almost zero.

Inulin used to measure GFR.
- ↳ filtered.
- ↳ not secreted, not absorbed.
- ↳ [inulin] in renal vein is still ↑ b/c only ~20% is filtered by glom.

$$\text{Clearance of inulin} = \frac{\text{Urine flow (mL/min)} \times [\text{urine inulin}]\ (\text{mg/mL})}{[\text{plasma inulin}]}$$

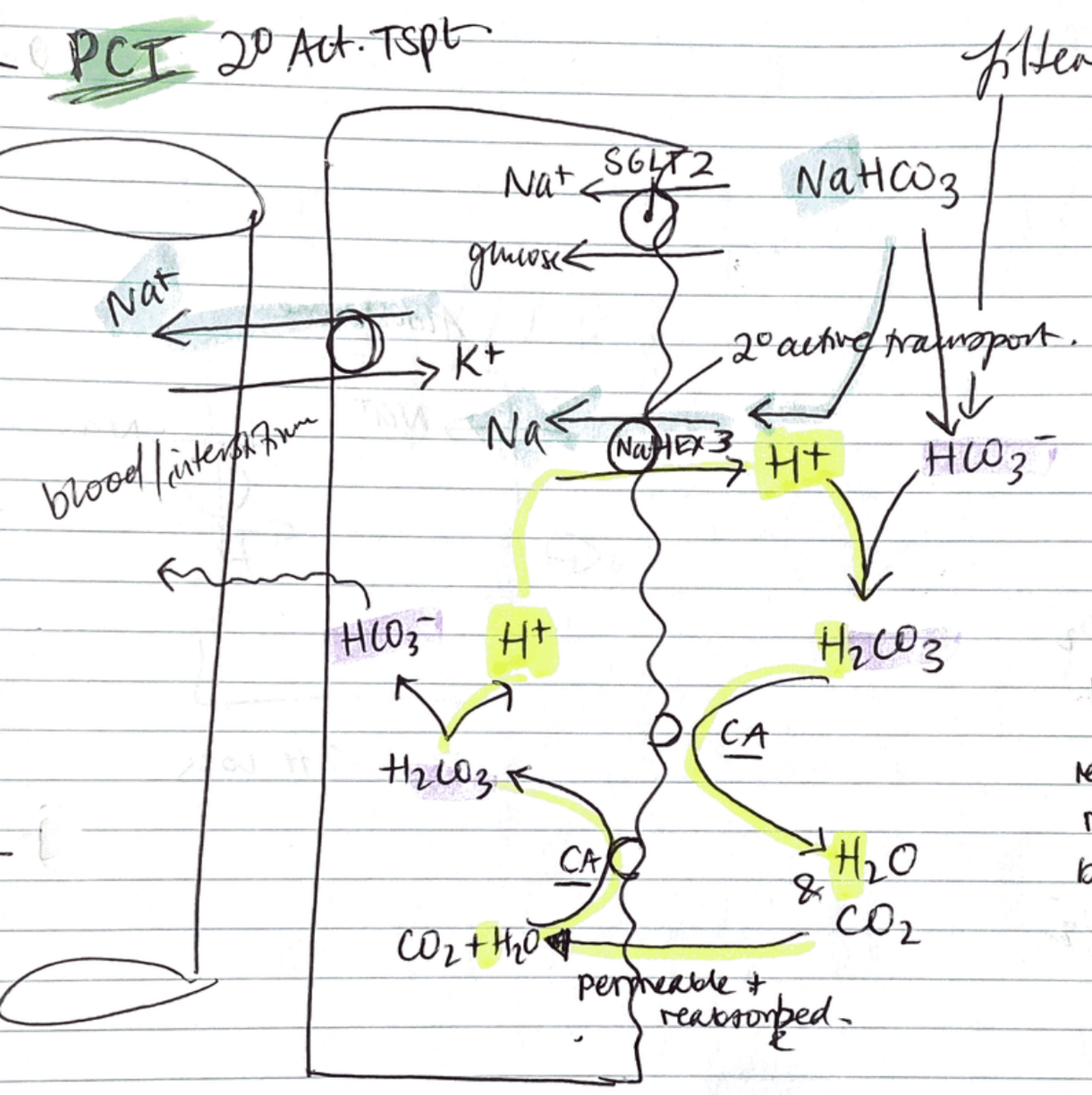
$$\text{PAH clearance (ERPF)} = \frac{\text{Urine flow} \times \text{Urine PAH}}{\text{Plasma PAH}}$$

$$\frac{\text{ERPF}}{0.9} = \text{Actual renal plasma flow}$$

$$\text{Renal Blood Flow} = \frac{\text{ARPF}}{1 - \text{HCT}}$$

H^+ & Acetazolamide

PCT 2° Act. TSpt



NORMALLY CA allows for recycling of H^+, resorption of bicarb and Na^+

prox. CT.

Acetazolamide ↓ CA activity ∴ ↓ Na resorption, ↓ K resorption distally via ↑ distal Na delivery → ↑ electrical potential that favours K^+ loss.

↑ urine pH from HCO_3^- loss can be seen in 30 mins. ↓ H^+ secretion can inhibit 85% of HCO_3^- resorption.

Causes → hyperchloraemic hypokalaemic metabolic acidosis. via ↑ NaCl reabsorption.

Used in Mt sickness → ↓ CSF formation & ↓ pH of CSF,

osmotic diuresis

Produced by compounds that are filtered but not absorbed. i.e. mannitol, polysaccharides
Also by naturally occurring substances that exceed resorption capacity.

{ Amount of water reabsorbed in proximal nephron is normal
  Maximal urine flow is ~~not~~ 16ml/min } WATER DIURESIS.

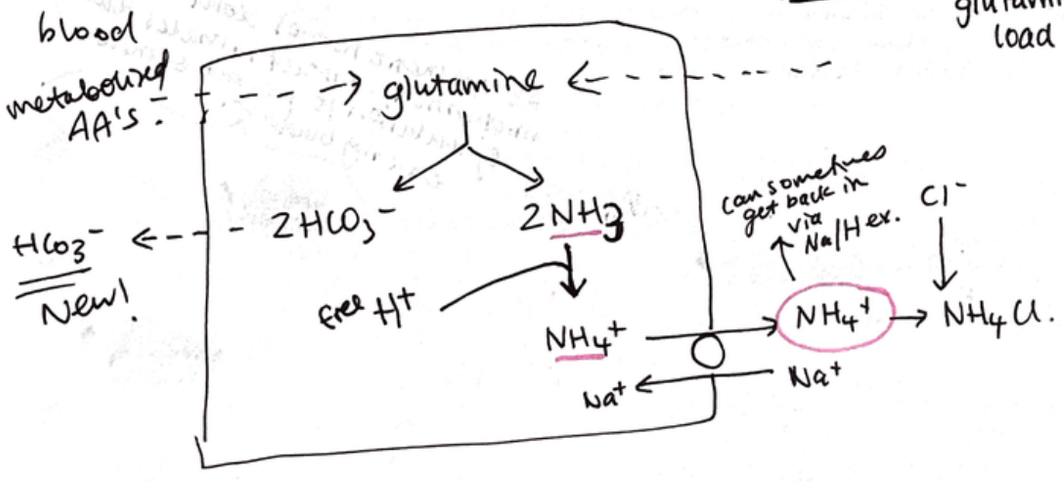
OSMOTIC DIURESIS → ↑ urine flow is 2° to ↓ reabsorption in proximal tubule / loops & very large urine flow can happen.

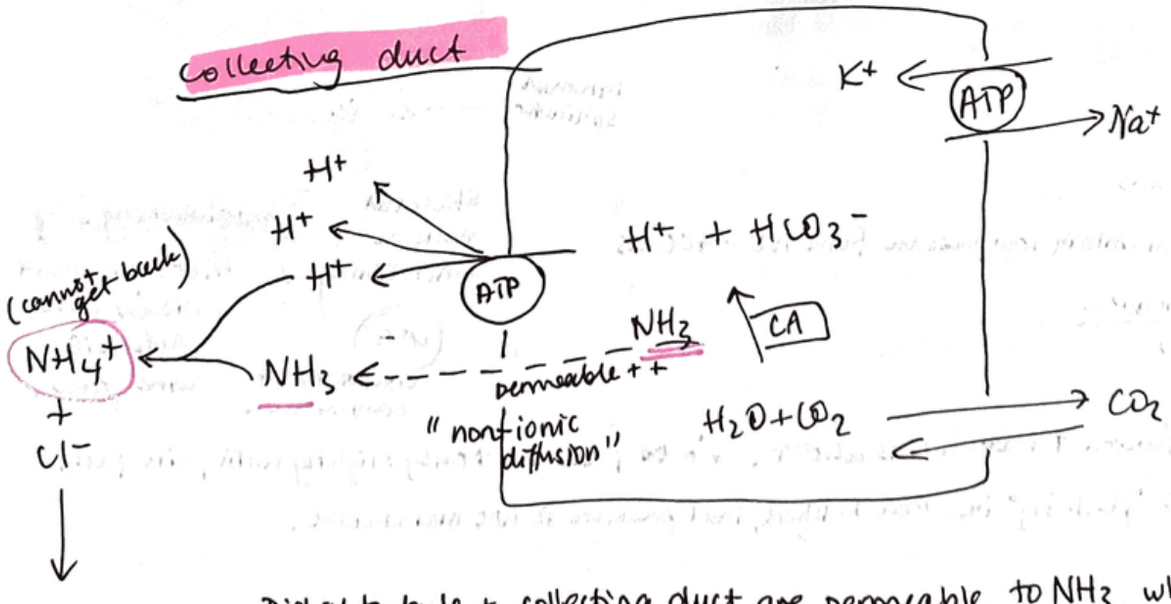
Secondary loss of electrolytes due to dilution.

Ammonia (NH_3) & Ammonium (NH_4^+) ⇒ new bicarb, buffering.

Proximal convoluted ~~collecting~~ tubule.

- glutamine synthesised in liver
- In PCT the breakdown of glutamine is enhanced by acid load and hypoK+





Distal tubule + collecting duct are permeable to NH_3, which quickly absorbs the free H^+ in the lumen.
These ducts are impermeable to NH_4^+ and so the extra H^+'s are trapped in the lumen